EXHIBIT D

Evaluation of Toxicology Claims Made Regarding Chemicals Attributed to the Rohm and Haas and Huntsman International LLC Facilities (the "Ringwood Site"), Ringwood, IL

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Professional Credentials

My name is Dr. Peter A. Valberg, and I am a public health professional specializing in human health risk assessment, toxicology, epidemiology, and biological modeling of human exposure to environmental chemicals, airborne gases, and airborne particles. My full *curriculum vitae* (CV) is attached as Exhibit A. For 23 years I was a faculty member in the Department of Environmental Health at the Harvard School of Public Health in Boston, Massachusetts. I am the author of 87 articles in peer-reviewed journals, plus many reports dealing with lung biology, airborne pollutants, and health effects. The CV at the end describes my credentials and includes a list of my publications.

I received a BA degree (*summa cum laude*) from Taylor University in 1964, a Ph.D. degree in physics in 1970 from Harvard University (as a student of Nobel laureate Prof. Norman F. Ramsey), and an M.S. degree in human physiology in 1975 from Harvard University.

My postdoctoral training included Biomedical Physics and Biomaterials Science (Massachusetts Institute of Technology, 1974), Alveolar Macrophage Function (National Heart, Lung, and Blood Institute, 1977), Pulmonary Pathology (University of Vermont Lung Center, 1979), Analytical and Quantitative Light Microscopy (Woods Hole Marine Biological Laboratory, 1984), and Advanced Quantitative Risk Assessment (University of Cincinnati, 1991).

I held the following academic research and training awards: Atomic Energy Commission Fellow (1964-67), Cottrell Research Corporation Science Grant (1972-75), National Science Foundation Fellow (1974-75), National Institutes of Health (NIH) New Investigator Pulmonary Research Award (1976-78), and Andrew W. Mellon Foundation Faculty Award (1981-1983).

I was a faculty member in the Physics Department of Amherst College from 1969-75. I was then hired by Harvard University. After spending two years as a Research Associate at the Harvard School of Public Health (HSPH), I was appointed in 1977 to the faculty of the Department of Environmental Health at HSPH. I was promoted to Associate Professor of Physiology in 1985, and remained on the faculty in the Department of Environmental Health for fifteen years. At HSPH, I directed and participated in research programs funded by the NIH, the titles of which are listed on my attached CV (Exhibit A).

As a faculty member in Harvard's Department of Environmental Health, I taught in part or in whole a wide variety of graduate and undergraduate courses on human physiology, airborne particles, and inhalation toxicology. These courses were taught at HSPH, as well as at Harvard College, Harvard Medical School, Massachusetts Institute of Technology, and Tufts University. The course titles included: Risk Analysis in Environmental and Occupational Health; Principles of Environmental Health; Human Physiology; Respiratory Physiology; Normal and Abnormal Human Physiology; Principles of Toxicology; Toxicology of Ambient Air Particles; Methods in Cell Biology; Structure and Function of the Mammalian Respiratory System; Indoor Air Quality; Aerosol Technology.

While a full-time faculty member at Harvard, I was invited to be a visiting scientist at other institutions on two occasions. In 1982, I was a Visiting Scientist at the Inhalation Toxicology Research Institute (ITRI) in Albuquerque, NM (the current name of this institute is the Lovelace Respiratory Research Institute). At ITRI, I designed and coordinated animal research that tracked the fate of substances inhaled into the body. In 1989, I was a Visiting Researcher at the Institute of Occupational Medicine in Helsinki, Finland. There, I participated in research on human subjects that examined the condition of their lung cells, as recovered by a lung lavage procedure. Throughout my research, teaching, and consulting career, I have been engaged in the study of the human health effects of chemical and physical agents in our environment.
I have served in an advisory capacity to a wide variety of governmental organizations concerned with health. These include the National Heart Lung and Blood Institute of NIH, the NIH Division of Research Grants, the Office of Health and Environmental Research (U.S. Department of Energy), the National Academy of Sciences, the U.S. Navy Office of Occupational Health and Preventive Medicine, the U.S. Department of Transportation, and the U.S. Environmental Protection Agency (USEPA). I also have been an advisor to the Health Effects Institute, the Center for Indoor Air Research, and the American Conference of Governmental Industrial Hygienists. For the Town of Needham (Massachusetts), I serve on the "Physical and Biological Sciences Study Committee," an environmental health assessment task force.

My health risk assessment work with Gradient Corporation began in 1990 while I was on the faculty at Harvard. Gradient is an environmental consulting company that provides health risk analyses to a variety of parties including government and industry. My work at Gradient involves the application of results from scientific research towards understanding the extent of human exposure to chemicals in the environment, as well as estimating the potential health effects of such exposures. As a Principal at Gradient, I provide expertise in risk assessment, human toxicology, and biological modeling of human exposure to environmental chemicals, airborne gases, and airborne particles. I have completed numerous projects

evaluating exposure and health effects of air pollutants, environmental chemicals, soil contamination, air toxics, volatile organic compounds (VOCs), and combustion emissions.

A number of the air-pollution projects that I have completed have been published and are listed on my CV. These include, for example:

1. An analysis of the levels at which inhalation exposure to the 189 Hazardous Air Pollutants (HAPs) identified in the 1990 Clean Air Act Amendments leads to noncancer toxicity. This report on the 189 HAPs was prepared for and published by the US EPA. [1]
2. A review of models by which airborne concentrations of diesel-exhaust particulate can be related to increments in lung cancer risk. The report was entitled, "Analysis of Diesel-Exhaust Cancer Potency Estimates derived from Animal Bioassays." [2]
3. An analysis of published studies of lung-cancer risk in workers exposed to elevated levels of airborne inert, insoluble particulate in occupational settings. The analysis examined whether lung cancer risk predicted from studies in laboratory rats exposed to overloading concentrations of inhaled, insoluble particles can be applied to human risk assessment. [3]
4. A meta-analysis of the lung-tumor response in rats inhaling airborne diesel exhaust particles over a wide range of concentrations. Our analysis revealed that the lung tumor response in rats exhibits a threshold level of exposure, below which an increased risk of lung tumors does not occur. [4]
5. An evaluation of the toxicological differences between carbon black and "soot." [5]
6. A review of occupational health issues in carbon-black manufacture, which was published in a standard text of occupational health, "Patty's Toxicology." [6]
7. A National Academy of Sciences review of the risk assessment methodologies that underlie USEPA's cost and benefit analyses for proposed air quality regulations. [7]
8. A health risk assessment for inhalation of respirable particles (PM_{10}) in ambient air. The assessment was based on chemical analyses of PM_{10} constituents and on toxicity factors for individual chemicals as developed by the US EPA for its Integrated Risk Information System data base. [8]

I am frequently selected by journal editors as a peer reviewer for toxicology articles submitted to many different journals, and these have included the *American Industrial Hygiene Journal, American Journal of Respiratory Cell and Molecular Biology, American Review of Respiratory Disease, Biorheology, Cell Biophysics, Critical Reviews in Toxicology, Environmental Health Perspectives, Environmental Science & Technology, Epidemiology, Experimental Lung Research, Fundamental and Applied Toxicology,*

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1 EPA/600/R-93/142
2 *Regulatory Toxicology & Pharmacology* 24:30, 1996
3 *Regulatory Toxicology & Pharmacology* 24:155, 1996
4 *Environmental Health Perspectives* 107:693, 1999
5 *American Journal of Industrial Hygiene Association Journal* 62:218, 2001
6 Patty's Toxicology, 5th Edition, E. Bingham, Ed., John Wiley & Sons NY, NY. 2001. Vol. 8, Ch. 111, Carbon Black
7 Estimating the Public Health Benefits of Proposed Air Pollution Regulations, National Academy Press, 2002
8 *Inhalation Toxicology* 16 (S1):19, 2004

Hepatology, Inhalation Toxicology, Journal of Aerosol Medicine, Journal of Applied Physiology, Journal of Applied Toxicology, Nature, Radiation Research, Risk Analysis, Science, and *Tissue & Cell*. I have also served on the editorial board of the Journal of Aerosol Medicine. During 2001-2002, I was invited by the National Research Council to serve on a National Academy of Sciences committee to analyze the risk-assessment basis of air quality regulations.

Outline of McCullom Lake Village Case

On behalf of Schnader Harrison Segal & Lewis, representing Rohm and Haas Chemicals and Huntsman International LLC (the "Ringwood Site"), I was asked to assess plaintiffs' toxicological claims being made regarding environmental contamination allegedly released by operations at the Ringwood Site in Ringwood, Illinois. For the work done in this case, Gradient Corporation bills my time at $290/hr. A list of cases where I have testified either at deposition or at trial in the past 4 years is provided as Exhibit B.

On April 25, 2006, plaintiffs filed a class action alleging, among other things, that residents of McCullom Lake Village had suffered exposure to environmental contamination, historical and current, in ambient air and in drinking water wells arising from "a [contaminated] groundwater plume running from the defendants' facilities onto Plaintiffs' and the [proposed] Class Members' properties," Complaint ¶ 105, Amended Complaint ¶ 127. Plaintiffs claim this alleged contamination has caused as many as sixteen alleged brain tumors in the McCullom Lake Village community and that the entire community is at increased risk of contracting brain cancer. Plaintiffs assert that one of the alleged contaminants, vinyl chloride (VC), is a "known cause of human brain cancer." [9]

I reviewed the Plaintiffs' 10/16/06 Motion for Class Certification, the 12/22/06 letter from plaintiffs' counsel to IEPA, and the 12/22/06 Plaintiffs' Opposition to Motion for Partial Summary Judgment, the relevant attached exhibits, as well as a number of other documents. The latter included the statements and deposition transcripts of Dr. Richard Neugebauer, Mr. James G. Hill, and Dr. Gary Ginsberg. I also reviewed the statement of plaintiffs' air-modeling expert, Dr. Paolo Zannetti.

I also have reviewed the reports of defendants' experts Dr. Peter J. Drivas and Mr. Robert D. Mutch.

I was asked to evaluate the toxicological basis for asserted claims of illness due to ambient air and drinking water exposure. Specifically, I was asked to apply the principles of human health risk assessment

[9] For example, Freiwald letter to IEPA, 12/22/06, p. 7

to determine whether the plaintiffs' claims of significant brain-cancer risk can be legitimately based on the toxicology of what is known about the chemicals at issue, and on what is known about their present and historical concentrations in McCullom Lake Village drinking water and ambient air.

Plaintiffs' Assertions Regarding Chemical Exposures and Health Consequences

Plaintiffs' toxicology expert, Dr. Gary Ginsberg, identifies in his statement [10] the substances of concern as trichloroethylene (TCE), 1,1-dichloroethylene (DCE) and vinyl chloride (VC).

Plaintiffs carried out domestic well testing of 7 residences in the McCullom Lake Village area (4/13/06), and fluoride was detected in a range from 0.07 to 0.53 mg/L, but there were no detects of the substances of concern in domestic well water. [11] In a separate series of tests of 9 private, residential wells in the McCullom Lake area, 1,1,1-trichloroethane (TCA) was detected in one private well at concentrations of 0.0015 and 0.0021 mg/L in two tests. [12] In none of these tests of domestic wells was a detectable level of TCE, DCE, or VC reported.

In addition to TCE, DCE, and VC, more recently, plaintiffs' groundwater expert, Mr. Hill, has claimed that the fluoride and TCA are site-related contaminants. [13] However, Mr. Hill stated in his deposition that he considered it unlikely that the TCA could have come from one of the defendants' facilities. [14]

Plaintiffs' groundwater expert, Mr. Hill, acknowledges that no evidence exists of current contamination in domestic wells in McCullom Lake Village. [15] Likewise, when asked about evidence for VC contamination in McCullom Lake Village wells in 1995 or 2000, Mr. Hill affirmed that there was no evidence of such contamination, [16] and went further to agree that he had no evidence today that any particular well in McCullom Lake Village had VC contamination at any particular time. [17] In fact, Mr. Hill acknowledged that he does not know the locations of the domestic wells in McCullom Lake Village, does

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10 Statement of Gary Ginsberg, 10/14/06
11 USBiosystems Analytical Results, Dated 4/21/06, provided by Layser & Freiwald, PC
12 Daniel E. Guif, McHenry Department of Health, letter of 6/15/06; Jennifer Davis, Illinois Department of Public Health, letter of 10/6/06
13 Additional affidavit of James Hill, 12/20/06
14 Hill Deposition, 11/27/06, p. 71:12.
15 Hill Deposition, 11/27/06, p. 60:13
16 Hill Deposition, 11/27/06, p. 61:12; p. 62:10
17 Hill Deposition, 11/27/06, p. 62:4

not know their depth, and does not know when they were installed. [18] Consequently, plaintiffs' experts have failed, by a large margin, to establish that any (let alone, sufficient) exposure to contaminated groundwater exists, or has existed.

Available Exposure and Dose Levels Are Below Toxicological Adverse-Health-Effect Thresholds

The words "toxic" and "hazardous" are not inherent properties of chemicals but require further qualification specifying how much, by what route of entry into the body, over what period of time, and so forth. Chemicals constantly enter the human body, whether found naturally in food, air, and water, or as synthetic chemicals from environmental exposures. Within the body, chemicals are transported, metabolized, and eliminated by various processes. As long recognized, from the 16th century Swiss physician, Paracelsus, to modern-day toxicologists: "The dose makes the poison." All substances are potentially toxic; it is the dose that differentiates a remedy, or a safe intake, from a toxic one. For any chemical, mere exposure does not automatically lead to an adverse effect. In this case, as in any other, the exposure concentrations, durations, and doses must be evaluated.

USEPA sets drinking water standards to assure that tap water in the US is safe to drink. One parameter is the Maximum Contaminant Level Goal (MCLG), the maximum level of a contaminant in drinking water at which no known or anticipated adverse effect on the health of persons would occur, and which allows an adequate margin of safety. MCLGs are non-enforceable public health goals. Once the MCLG is determined, USEPA sets an enforceable standard. This standard is a Maximum Contaminant Level (MCL), the maximum permissible level of a contaminant in water which is delivered to any user of a public water system, and the MCL is set as close to the MCGL as feasible.

For the chemicals mentioned above, the following table shows the drinking-water MCL and MLCG values, [19] along with the concentrations reported in tests of McCullom Lake Village drinking water from private wells. The reported concentrations are far below the applicable MCL's and MCLG's.

[18] Hill Deposition, 11/27/06, p. 77-78

[19] http://www.epa.gov/safewater/contaminants/index.html#mcls

Substance	**Drinking Water MCL** (mg/L)	**Drinking Water MCLG** (mg/L)	**Drinking Water Measurements** (mg/L)
TCE	0.005	zero	none detected
1,1,1-TCA	0.2	0.2	0.0015 – 0.0021
1,1-DCE [20]	0.007	0.007	none detected
VC	0.002	zero	none detected
Fluoride	4	4	0.07 – 0.53

Also, recent sampling of three groundwater monitoring wells installed immediately south of the Ringwood Site (*i.e.*, on the south side of the site, which faces McCullom Lake Village) over a range of groundwater depths (66 ft to 232 ft) showed non-detect concentrations (below 0.001 mg/L) for all the above volatile organic compounds, namely TCE, TCA, DCE, and VC. [21] These results support the lack of any groundwater contamination moving southward from the Ringwood Site toward McCullom Lake Village.

In addition to actual groundwater measurements, understanding groundwater movement supports the view that the chemicals of concern have not reached plaintiffs' drinking-water wells. Defendants' hydrogeology expert, Robert Mutch, has demonstrated that "The well documented and thoroughly studied groundwater contamination associated with the Rohm and Haas site poses no risk to residents of McCullom Lake Village." [22]

Plaintiffs' toxicology expert, Dr. Ginsberg, acknowledges that it is unknown whether there have been any releases of industrial chemicals into McCullom Lake Village drinking water, and that it is unknown whether there have been any VC exposures to these residents. [23] Dr. Ginsberg acknowledges that the exposure could be zero. [24]

For airborne, non-carcinogenic chemicals, the health-protective level is called a "reference concentration," or RfC, which is the concentration of continuous, lifetime inhalation exposure that is not expected to result in adverse health effects. For carcinogenic chemicals the "unit risk," or UR is the

[20] MCL and MCLG for isomers of DCE (cis-1,2-DCE; trans-1,2-DCE) are about ten-fold larger than for 1,1-DCE
[21] URS Report of December 2006, Tables 2 and 3
[22] Expert Report of Robert D. Mutch, January 17, 2007, p. 4-1
[23] Ginsberg Deposition, 1/4/07, p. 161:24; p. 174:1-10
[24] Ginsberg Deposition, 1/4/07, p. 165:8-9

concentration of continuous, lifetime inhalation exposure that leads to a 1-in-a-million lifetime cancer risk, assuming a default, linear extrapolation of risk to zero concentration. The 1-in-a-million lifetime cancer risk is a very stringent *de minimus* risk level that should be compared to the normal, lifetime cancer risk of everyone, which is approximately 418,000-in-a-million. [25] The normal, lifetime risk for brain cancer for everyone is approximately 5,800-in-a-million. [26]

Using the inventory of air emissions from Rohm and Haas (1988-2004), as well as information on emissions from the air stripper (1992-2000), and concentrations of contaminants in the groundwater plume (1996-2004), Dr. Peter Drivas predicted that plaintiffs' possible airborne exposures to DCE and VC from the simultaneous operation of these sources were very low. [27] The air modeling of Dr. Drivas predicted outdoor air concentrations of DCE and VC in the McCullom Lake Village area, with DCE annual average concentrations of 0.06 micrograms per cubic meter ($\mu g/m^3$) and VC annual average concentrations of 0.0003 $\mu g/m^3$. [28] These levels are far below the reference concentration (RfC) for DCE, which is 200 $\mu g/m^3$, and the 1-in-a-million lifetime cancer risk concentration for VC, which is 0.23 $\mu g/m^3$ (which is set based on the most sensitive health endpoint, liver angiosarcomas).

Substance	**No-Adverse-Health-Effect (or *de minimis*) Benchmarks,** [29, 30] **$\mu g/m^3$**	**Outdoor Air Concentration Predicted** [31] **for McCullom Lake Village, $\mu g/m^3$**
1,1-DCE	200	0.06
VC	0.23	0.0003

As already noted, background cancer risks are far higher than calculated hypothetical risks from the VC air levels predicted by Dr. Drivas. Assuming that VC causes cancer at very low levels of exposure, and using the unit risk factor provided by the United States Environmental Protection Agency (USEPA), I calculated the hypothetical cancer risk due to inhaling the modeled air concentration of VC in the Table above. For an assumed 70-year exposure duration, the result is 0.001-in-a-million (or 1-in-a-billion), which essentially means that for a single individual continuously inhaling this concentration, one cancer might be expected to occur every 70 billion years (every 70,000,000,000 years). This calculated risk can also be

25 American Cancer Society http://caonline.amcancersoc.org/cgi/content/full/56/2/106
26 Central Brain Tumor Registry of the U.S., 2006, "Primary Brain Tumors in the U.S., Statistical Report, 1998-2002"
27 Dr. Peter J. Drivas expert report of January 22, 2007
28 Dr. Peter J. Drivas expert report of January 22, 2007
29 USEPA: http://cfpub.epa.gov/iris/quickview.cfm?substance_nmbr=0039
30 USEPA: http://cfpub.epa.gov/iris/quickview.cfm?substance_nmbr=1001
31 Dr. Peter J. Drivas expert report of January 22, 2007, Table 1, p. 2

expressed as one cancer in a population of one million people every 70,000 years. That is, this hypothetical, but conservatively calculated, risk is essentially zero.

The Ringwood Site includes the "TPU unit," which is now operated by Huntsman International, LLC, and has been since 2000. The Illinois Hazardous Air Pollutant (HAP) air-emissions summary for Huntsman reports minor air emissions, which do not include TCE, DCE, or VC. [32] None of the air emissions are chemicals classified as carcinogenic. Therefore, I will not discuss the toxicology of the air emissions attributed to Huntsman.

The statement of plaintiffs' air expert, Dr. Zannetti, provides no air dispersion modeling in support of the plaintiffs' claim that airborne vapors of the chemicals of concern can migrate in any significant quantity toward plaintiff homes. [33]

Thus, based both in terms of what is known about drinking water exposure and about ambient air exposure, exposure levels are, and have been, far below thresholds of concern for adverse health effects.

Applying generally accepted methods of toxicology, *e.g.*, "the dose makes the poison," and relying on the concentrations of the chemicals of concern (identified above) that have been estimated or measured for ambient air and drinking water by experts in this case, I am of the opinion that the plaintiffs' claim regarding exposure to the chemicals of concern causing as many as sixteen excess alleged brain tumors in the McCullom Lake Village community is not supported by the available environmental concentration, exposure, and toxicology evidence.

The Magnitudes of Any Potential Doses of Substances of Concern Would Be Heterogeneous

Setting aside the lack of evidence for any actual exposure, it's clear that people drink different amounts of tap water and breathe differing amounts of outdoor air. Thus, even if there were chemicals of concern in water or air, the members of the proposed class (*i.e.*, all the past and present residents of McCullom Lake Village) would experience widely heterogeneous amounts of ambient air and groundwater (drinking) exposure. Differences in exposure lead to varied inhaled and ingested doses to such a degree that all residents cannot be considered as experiencing a "typical health risk" over the proposed class-wide basis.

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[32] Dr. Peter J. Drivas expert report of January 22, 2007

[33] Statement of Paolo Zannetti, 10/12/06

The expected variability regarding amounts and routes of exposure means that a dose evaluation for one person cannot be applied to others. Mr. Hill, the plaintiffs' groundwater expert, acknowledges that any potential contamination among domestic wells would differ from home to home both in time period, duration, and level of contamination. [34]

The drinking-water exposure of each resident would vary widely not only because the duration and levels of well-water contamination would be different, but also because the amount of domestic-well tap water actually consumed depends on many individual factors. First, one would need to determine what fraction of time is spent at home *versus* away at the office, at school, shopping, visiting, *etc.* Then, one would need to determine how much fluid intake comes from tap water as opposed to all other sources of ingested water (for example, bottled water, milk, juice, sodas, beer, and any other fluid-containing foodstuffs). Finally, the overall fluid intake rate for an individual depends on body type, lifestyle, and exercise / activity patterns. It can be expected that careful evaluation of these factors would demonstrate that the ingested dose for each plaintiff is different.

As in the case of drinking water, the exposure of residents to any outdoor-air chemicals would vary widely not only because both the duration of contact and the outdoor-air levels would be different for each person, but also because the amount of outdoor air actually inhaled depends on many individual factors. First, one would need to determine what fraction of time is spent at home *versus* away at the office, at school, shopping, visiting, *etc.* Then, one would need to determine the fraction of time that is spent outdoors *versus* indoors, because the concentrations in indoor air will be different from those in outdoor air. Finally, the overall volume of air breathed each day depends on body type, lifestyle, and exercise / activity patterns. It can be expected that careful evaluation of these factors would demonstrate that the inhaled dose for each plaintiff is different.

Not only would any potential exposure concentrations, intake behavior, and doses be heterogeneous among a group of people, but also, as plaintiffs' expert Dr. Ginsberg stated, if any exposure were to occur, then intake, metabolism, and excretion of chlorinated solvents will be different in different people, as will detoxification and DNA repair. [35] Dr. Ginsberg is not planning to quantify risk on an individual basis. [36]

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[34] Hill Deposition, 11/27/06, p. 91:1; p. 108:23; p. 145:1-7; p. 146:20-22; p. 243:19

[35] Ginsberg Deposition, 1/4/07, p. 31:2-9; p. 59:3-16; p. 230:13-17 and p. 231:23-25

[36] Ginsberg Deposition, 1/4/07, p. 136:16-18; p. 233:11-16

Chemical metabolism (activation and deactivation) and chemical excretion show a high degree of variability among individuals. [37] Likewise, susceptibility to DNA damage and DNA repair ability are different for different individuals, [38] and this has been specifically shown in relation to repair of DNA damage related to VC exposure. [39, 40] Hence the susceptibility of individuals in the proposed plaintiff class to the potential cancer risk of a given amount of VC would differ widely.

Plaintiffs' epidemiology expert, Dr. Neugebauer, stated that, even though the average latency period (time between first exposure and disease manifestation) for glioblastoma is 21 years, there is likely a "distribution of latency among individuals non-occupationally exposed," meaning that the latency period assigned to one person cannot be assumed typical of all proposed class members. [41] Moreover, Dr. Ginsberg acknowledges that he does not even know the times of residence that apply to the McCullom Lake brain cancer cases. [42]

Dr. Neugebauer acknowledges that, aside from any potential groundwater contamination, different people in the community will have individualized baseline risk factors for brain cancer. [43] He stated that there are different factors that "influence a person's risk for developing [cancer]." [44] That is, the suspected risk factors for glioblastoma and oligodendroglioma would be expected to exhibit significant heterogeneity among the proposed class members. As noted before, Dr. Ginsberg states that he will not be looking at individual risk factors. [45]

In summary, plaintiffs' experts have not established any homogeneity of exposure to the chemicals of concern, or any homogeneity of brain cancer risk, among the members of the proposed class.

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37 Klaassen, CD. 2001. Casarett and Doull's Toxicology: The Basic Science of Poisons (Sixth Edition) McGraw-Hill (New York) p. 26 and 99.

38 Bartsch H, Hietanen E. 1996. The role of individual susceptibility in cancer burden related to environmental exposure. *Environ Health Perspectives* 104 Suppl 3:569-77

39 Zhu S, Wang A, Xia Z. 2005. Polymorphisms of DNA repair gene XPD and DNA damage of workers exposed to vinylchloride monomer. *Int J Hyg Environ Health* 208:383-90

40 Li Y, Lee S, Marion MJ, Brandt-Rauf PW. 2005. Polymorphisms of microsomal epoxide hydrolase in French vinyl chloride workers. *Int J Occup Med Environ Health* 18:133-8

41 Neugebauer Deposition, 11/21/06, p. 186:2-4

42 Ginsberg Deposition, 1/4/07, p. 28:22-25

43 Neugebauer Deposition, 11/21/06, p. 238:6

44 Neugebauer Deposition, 11/21/06, p. 214:21-25

45 Ginsberg Deposition, 1/4/07, p. 136:16-18; p. 233:11-16

Causal Link Between Alleged Exposures and Brain Cancer Risk Has Not Been Established

Setting aside the lack of evidence for actual exposure, and setting aside the issue of heterogeneity of putative risk among the members of the proposed class, generally accepted scientific evidence does not establish that the brain cancers at issue here are caused by the identified chemicals of concern.

From the materials I have reviewed, as provided by plaintiffs' experts, Dr. Ginsberg, Mr. Hill, Dr. Neugebauer, and Dr. Zannetti, none of the following questions regarding causation have been affirmatively answered so as to establish a link between the members of the proposed class and brain-cancer risk:

1. Do worker populations exposed (at higher levels than residents) to DCE, TCE, TCA, VC, or fluoride develop glioblastoma or oligodendroglioma as a result of such exposures?
2. Did the any of the members of the proposed class have contact with any of the chemicals of concern in such a way as to receive doses of magnitude sufficient to cause adverse health effects, and if so, which ones?
3. Was the timing of the putative chemical exposure related *via* an appropriate latency period to the diagnoses of glioblastoma or oligodendroglioma?
4. Have hypothesized, alternative causes of the glioblastomas and oligodendrogliomas been ruled out?
5. Do animal studies reliably confirm that any of the above chemicals lead to malignant neoplasia in astrocytes or oligodendrocytes?

The answers to each of the above questions, all of which need to be affirmative in order to support the allegations, are in fact negative.

The risk for glioblastoma or oligodendroglioma is not reported to have any relation to DCE, TCE, TCA, and fluoride exposure. [46] In his statement and deposition, plaintiffs' expert Dr. Ginsberg focuses on VC exposures as a cause of glioblastoma and oligodendroglioma.

Dr. Ginsberg's statement and deposition note some early studies that explored a possible link between brain cancer risk and VC exposure in polymerization workers exposed to high levels of VC monomer in the process of polyvinylchloride (PVC) production. However, Dr. Ginsberg does not cite any of the more recent reviews of the literature. These in-depth summaries and updates of worker cohorts do not support a causal link between VC exposure and brain cancer, and do not support cancers being caused in

[46] El-Zein *et al*. 2005. "Brain Cancer," Chapter 25 in Cancer Risk Assessment, P.G. Shields, Ed., Taylor and Francis, Boca Raton, FL

residential populations living a mile away from facilities such as those belonging to the defendants (Mundt *et al*. 2000 [47]; Ward *et al*. 2001 [48]; Bosetti *et al*. 2003 [49]; Lewis and Rempala, 2003 [50]; IRIS, 2003 [51]; El-Zein *et al*., 2005 [52]; ATSDR, 2006 [53]).

The scientific literature indicates that the "most sensitive site" for cancer risk from elevated VC exposures is liver angiosarcoma. [54] But, plaintiffs' claims here are based entirely on a purported brain tumor cluster, a connection not supported by the scientific literature. Despite the lack of support for such a connection, Dr. Ginsberg postulates that brain cancers were not prevalent in studies of VC-exposed workers because the liver angiosarcomas killed them first. He states "the liver's chance of getting tumors, and therefore decrease your chance of seeing it in the brain, because these workers are going to die of liver cancer first." [55] I am not aware of any occupational health authority, scientific consensus document, or peer-reviewed research paper that espouses the idea that VC-exposed workers who developed liver angiosarcoma were destined to develop glioblastoma or oligodendroglioma, had they survived liver angiosarcoma. However, Dr. Ginsberg claims that this conclusion "drops right out of the literature." [56]

In his book, "What's Toxic, What's Not," Dr. Ginsberg states that "VOCs (benzene, TCE, PERC, *etc.*) are widespread in consumer products and gasoline" and "there are many sources of VOCs indoors." [57] Even though common VOCs like benzene and 1,3-butadiene are considered human carcinogens, Dr. Ginsberg does not describe any consideration of evaluating plaintiffs' possible exposures to benzene and 1,3-butadiene. Also, Dr. Ginsberg has not considered assessing VC from sources other than what is allegedly attributed to the defendants, *e.g.*, he seemed unaware that VC in drinking water can derive from supply pipes made of PVC. [58]

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47 Mundt KA, Dell LD, Austin RP, Luippold RS, Noess R, Bigelow C. 2000. Historical cohort study of 10 109 men in the North American vinyl chloride industry, 1942-72: update of cancer mortality to 31 December 1995. *Occup Environ Med*. 57:774-81

48 Ward E, Boffetta P, Andersen A, Colin D, Comba P, Deddens JA, De Santis M, Engholm G, Hagmar L, Langard S, Lundberg I, McElvenny D, Pirastu R, Sali D, Simonato L. 2001. Update of the follow-up of mortality and cancer incidence among European workers employed in the vinyl chloride industry. *Epidemiology* 12:710-8.

49 Bosetti C, La Vecchia C, Lipworth L, McLaughlin JK. 2003. Occupational exposure to vinyl chloride and cancer risk: a review of the epidemiologic literature. *Eur J Cancer Prev*. 12:427-30

50 Lewis R, Rempala G. 2003. A case-cohort study of angiosarcoma of the liver and brain cancer at a polymer production plant. *J Occup Environ Med*. 45:538-45

51 U.S. Environmental Protection Agency, Integrated Risk Information System: http://www.epa.gov/iris/subst/1001.htm

52 El-Zein *et al*. loc. cit.

53 Agency for Toxic Substances & Disease Registry (ATSDR), Centers for Disease Control (CDC) : http://www.atsdr.cdc.gov/toxprofiles/tp20.html

54 USEPA, IRIS, loc. cit.

55 Ginsberg Deposition 1/4/07, p. 245:10-13

56 Ginsberg Deposition 1/4/07, p. 248:2-3

57 G. Ginsberg and B. Toal, What's Toxic and What's Not. Berkeley Publishing Group, New York, 2006. pp. 177-161

58 http://www.dnr.mo.gov/pubs/pub2123.pdf

Dr. Ginsberg has not defined what dose of VC would be required for plaintiffs in the proposed class to reasonably make these individuals at significant risk for glioblastoma or oligodendroglioma. That is, any risk assessment calculation must recognize that some doses may lead to unacceptable risk, but sufficiently small doses are without reasonable expectation of adverse effects.

Dr. Ginsberg merely mentions age, gender, ethnicity, and income as modifiers of brain cancer risk, and he does not sufficiently address many hypothesized risk factors for brain cancer. [59] Although Dr. Ginsberg acknowledges ionizing radiation and N-nitroso compounds (nitrosamines), he does not consider other, albeit hypothetical, brain cancer risk factors (El-Zein *et al*., 2005 [60]; Wrensch *et al*., 2002 [61]) such as electromagnetic fields, cell phones, coffee, smoking, cured foods, asthma, [62] serum cholesterol levels, virus exposure, pesticides, lead (Pb) contamination, [63] PCB contamination, [64] and medications. [65] Dr. Ginsberg's statement neither describes looking into these other possible influences on brain cancer rates. Nor does Dr. Ginsberg discuss ionizing radiation exposure (which he acknowledges may be linked to brain cancer) from environmental sources, *e.g.* radon in air and radium in drinking water, both of which occur in this area of Illinois. [66]

Dr. Richard Neugebauer, plaintiffs' epidemiology expert, states that he has yet to do a literature search to "identify the chief factors that increased or influenced the persons risk for developing [glioblastoma or oligodendroglioma]." [67] Thus, Dr. Neugebauer has not developed the data necessary to identify individual risk factors and to rule out alternative causes for the two brain cancer types he will be analyzing. Dr. Neugebauer likewise does not know what latency period exists between potential VC exposure and disease manifestation for these cancers. [68] Finally, Dr. Neugebauer states that he has not yet determined whether epidemiology evidence supports a risk for both inhalation and ingestion exposure

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59 Ohgaki H, Kleihues P. 2005. Epidemiology and etiology of gliomas. *Acta Neuropathol (Berl)*. 109:93-108

60 El-Zein *et al*. loc. cit.

61 Wrensch M, Minn Y, Chew T, Bondy M, Berger MS. 2002. Epidemiology of primary brain tumors: current concepts and review of the literature. *Neuro-oncology* 4:278-99

62 Schoemaker MJ, Swerdlow AJ, Hepworth SJ, McKinney PA, van Tongeren M, Muir KR. 2006. History of allergies and risk of glioma in adults. *Int J Cancer* 119:2165-72

63 van Wijngaarden E, Dosemeci M. 2006. Brain cancer mortality and potential occupational exposure to lead: findings from the National Longitudinal Mortality Study, 1979-1989. *Int J Cancer* 119:1136-44

64 Ruder AM, Hein MJ, Nilsen N, Waters MA, Laber P, Davis-King K, Prince MM, Whelan E. 2006. Mortality among workers exposed to polychlorinated biphenyls (PCBs) in an electrical capacitor manufacturing plant in Indiana: an update. *Environ Health Perspectivews* 114:18-23

65 Houben MP, Coebergh JW, Herings RM, Casparie MK, Tijssen CC, van Duijn CM, Stricker BH. 2006. The association between antihypertensive drugs and glioma. *Br J Cancer* 94:752-6

66 http://www.tmcnet.com/usubmit/2006/12/08/2152251.htm

67 Neugebauer Deposition, 11/21/06, p. 214:18-23

68 Neugebauer Deposition, 11/21/06, p. 185:20-21

pathways. [69] Consequently, the ability to assert that all the members of the proposed class are at uniformly increased risk is severely compromised.

Although Dr. Ginsberg cites some early laboratory-animal studies that had equivocal implications regarding brain cancers, one of the exhaustive, more recent animal experiments concluded that "There was no evidence that feeding of VCM [vinyl chloride monomer] affected the incidence of tumors in organs other than the liver." [70]

In summary, plaintiffs' experts have not established that exposure to the chemicals attributed to defendants' facilities can increase the rate of glioblastomas and oligodendrogliomas among the members of the proposed class.

The Medical Monitoring Has Not Been Justified as to Need, Design, and Beneficial Results

Setting aside all the deficiencies in the plaintiffs' claims – – the lack of evidence for actual exposure, the issue of heterogeneity of putative risk among the members of the proposed class, and the lack of scientific evidence that the brain cancers at issue are caused by the chemicals of concern, – – no explanation is given for how the medical monitoring would benefit the members of the proposed class.

The plaintiffs' request for medical monitoring requires evidence for sufficient risk of future disease in individuals having currently no sign or symptom of the disease. The following criteria have been adopted by the Agency for Toxic Substances and Disease Registry (ATSDR) as requirements, all of which must be satisfied, before a medical monitoring program can be deemed necessary: [71]

1. Documented evidence of exposure at a sufficient level of risk, *i.e.*, the population being screened must be at significantly higher risk for the undiagnosed disease such that the screening will not be merely detecting background prevalence in the population;
2. A population at risk that is well-defined and can be identified;
3. A scientific basis exists for an association between exposure and health effects;
4. The health effects are detectable and amenable to prevention/intervention, *i.e.*, early detection through screening should be known to have an impact on the natural history of the disease process (*i.e.*, glioblastoma and oligodendroglioma);

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69 Neugebauer Deposition, 11/21/06, p. 187:3-5

70 Til HP, Feron VJ, Immel HR. 1991. Lifetime (149-week) oral carcinogenicity study of vinyl chloride in rats. *Food Chem Toxicol*. 29:713-8.

71 http://www.atsdr.cdc.gov/testimony/testimony-1998-09-16.html

5. Medical screening requirements are satisfied, *i.e.*, there must be an accepted screening test that meets the requirements for validity, reliability, estimates of yield, sensitivity, specificity, and acceptable cost;
6. Accepted treatment/intervention exists and a referral system is available; and
7. Logistics must be resolved prior to program implementation.

The materials provided by the plaintiffs' experts do not address any of these points. The toxicological profiles of the chemicals at issue do not support the idea that they would cause increases in glioblastoma and oligodendroglioma risk.

Surveillance screening for neuroblastoma of asymptomatic individuals is not an established procedure. [72, 73, 74] A major 5-year, neuroblastoma screening study, *i.e.*, "Quebec Neuroblastoma Screening Project" (QNSP) determined that "screening did not decrease neuroblastoma mortality, and caused adverse health effects." Furthermore, the authors determined that the money spent on QNSP was worthwhile, not because it improved neuroblastoma prognosis, but rather, because the QNSP "evaluation averted at least 14 years of ineffective screening across North America, thereby preventing important adverse health effects [due to screening] and much wasteful health spending." [75]

In summary, the medical monitoring program has not been justified by plaintiffs' experts, in terms of the need, the procedures used, and the expected beneficial results.

Overall, the plaintiffs' claims fail on many grounds, any one of which is a serious deficiency, namely, (a) the absence of any actual (documented or modeled) exposure of meaningful magnitude, (b) the issue of heterogeneity of putative risk among the members of the proposed class, (c) the lack of scientific evidence that the brain cancers at issue are caused by the chemicals of concern, and (d) no substantiation of how the medical monitoring would benefit the members of the proposed class.

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72 Woods WG, Tuchman M, Robison LL, Bernstein M, Leclerc JM, Brisson LC, Brossard J, Hill G, Shuster J, Luepker R, Byrne T, Weitzman S, Bunin G, Lemieux B, Brodeur GM. 1997. Screening for neuroblastoma is ineffective in reducing the incidence of unfavourable advanced stage disease in older children. *Eur J Cancer* 33:2106-12

73 http://bms.brown.edu/cme/files/journalcme/cancerupdate.pdf

74 Spix C, Michaelis J, Berthold F, Erttmann R, Sander J, Schilling FH. 2003. Lead-time and overdiagnosis estimation in neuroblastoma screening. *Stat Med*. 22:2877-92

75 Soderstrom L, Woods WG, Bernstein M, Robison LL, Tuchman M, Lemieux B. 2005. Health and economic benefits of well-designed evaluations: some lessons from evaluating neuroblastoma screening. *J Natl Cancer Inst*. 97:1118-24

Exhibit A

Gradient
CORPORATION

20 University Road
Cambridge, MA 02138
617-395-5000

Peter A. Valberg, Ph.D.
Principal

Areas of Expertise

Public health, inhalation toxicology, epidemiology, human health risk assessment, risk communication, indoor / outdoor air quality, comparative toxicology, modeling of human exposure and retained dose, health effects of ionizing and non-ionizing radiation.

Education

M.S., Human Physiology and Inhalation Toxicology, Harvard School of Public Health.

Ph.D., Physics, Harvard University, Graduate School of Arts and Sciences.

M.A., Physics, Harvard University.

A.B., Physics and Mathematics, *summa cum laude*, Taylor University.

Professional Experience

2001 – Present (and 1990 – 1998) GRADIENT CORPORATION, Cambridge, MA
Principal. Environmental consulting practice includes inhalation toxicology; environmental health; human health risk assessment; use of epidemiology in public health decisions; health effects of airborne gases and particles; health effects of ionizing and non-ionizing radiation.

1998 – 2000 CAMBRIDGE ENVIRONMENTAL, INC., Cambridge, MA
Senior Scientist

1985 – 2000 HARVARD SCHOOL OF PUBLIC HEALTH, Boston, MA
Associate Professor of Human Physiology. (Adjunct, after 1990) Research work included: (1) human health effects of air toxics, (2) lung macrophage function measured with magnetic particles, (3) lung deposition and clearance of radioactive tracer particles.

1989 INSTITUTE OF OCCUPATIONAL HEALTH, Helsinki, Finland
Visiting Researcher. Developed a magnetometric assay to be used for studying pulmonary macrophage function for lung cells lavaged from human subjects.

1982 INHALATION TOXICOLOGY RESEARCH INSTITUTE, Albuquerque, NM
Visiting Scientist. Examined the effect of exercise and hypercapnia on deposition, lung clearance, and lung distribution of inhaled radioactive aerosol.

1976 – 1985 HARVARD SCHOOL OF PUBLIC HEALTH, Boston, MA
Assistant Professor of Respiratory Physiology.

1970 – 1976 AMHERST COLLEGE, Amherst, MA
Assistant Professor of Physics.

Professional Activities

- National Academy of Sciences and National Research Council, Evaluating Health-Risk-Reduction Benefits of USEPA Regulations (2001 – 2003)
- Harvard School of Public Health: Research Advisory Committee Member for NIH-Sponsored Research on "Mechanisms of mortality/morbidity due to air particulate" (1997 – 2006)
- Member of the Committee on Man and Radiation (COMAR) (1999 – 2006)
- Health Effects Institute, Cambridge, MA, *ad hoc* reviewer (1984 – 94)
- National Research Council, Commission on Life Sciences: Committee on Passive Smoking (1986-88)
- Editorial Board, *Journal of Aerosol Medicine* (1987- 2000)
- Center for Indoor Air Research, grant-application reviewer (1989 – present)
- NIOSH: Environmental Center Grants, Site Visit Delegation (1990)
- NIH Reviewer: Cardiovascular and Pulmonary Study Section, Radiation Study Section, and Health of the Population Study Section
- DOE: Office of Health and Environmental Research, reviewer
- Harvard Center for Risk Analysis: Peer Review Board on Cellular Telephones (1994 – 99)
- Physical and Biological Sciences Study Committee, Town of Needham Planning Board

Professional Affiliations

Society of Toxicology (full member) • International Society for Environmental Epidemiology • Society for Risk Analysis • Health Physics Society (full member) • International Society for Aerosols in Medicine • Sigma Xi • American Association for the Advancement of Science

Projects *(abbreviated)*

Carbon Black Manufacturers: Evaluated the toxicology and epidemiology of carbon black inhalation and ingestion.

City of Newton Health Department: Measured RF levels from a local transmitting antenna, reviewed RF field calculations, and provided scientific literature critique on RF health effects.

Confidential Client: Prepared a risk assessment for a Massachusetts landfill containing both chemical and radioactive waste and including multiple pathways of contaminant uptake by a trespasser.

Confidential Client: Prepared a model predictive of asbestos fiber drift and inhalation health hazard applicable to industrial processes where asbestos-containing materials are used.

Confidential Clients: Prepared an analysis of relative risks of TCE in drinking water *versus* health hazards from background levels of chemicals in air, water, and soil, as well as other routine risks to life and health.

Confidential Clients: Prepared human toxicology profiles for a range of chemical substances, including beryllium, carbon black, chlorine, coke oven emissions, copper, ferrocene, Freon, manganese, *n*-butylamine, and thorium.

Electric-Power Generating Companies: Prepared and delivered expert reports and public testimony on the potential health effects of airborne emissions from coal fired, gas fired, oil fired, and wood-fired electric utility power generating plants.

Electric Power Research Institute: Reviewed and analyzed the mechanisms by which biological systems may be affected by environmental electric and magnetic fields (EMFs). Organized a workshop on the causes and characteristics of childhood leukemia.

Engine Manufacturers Association: Prepared critiques of the U.S. EPA and California EPA health assessment documents on the potential carcinogenicity of diesel exhaust and ambient air particulate matter.

Harvard School of Public Health: Continuing Education for Professionals: Prepared material on special topics on inhalation toxicology for graduate students and health professionals: Presented lectures on risk assessment and risk communication. Presented case studies on health risks of electric and magnetic fields and cellular telephones.

Health Effects Institute: Prepared an analysis entitled "Ozone Molecular Dosimetry and Interaction with Biological Macromolecules."

Health Effects Institute: Organized, supervised, and documented a feasibility study for the Health Effects Institute initiating a national research program on the health effects of electric and magnetic fields.

Manufacturing Company: Analyzed multi-pathway human health risk for a site contaminated with polychlorinated biphenyls (PCBs) and chlorinated organic solvents. Analyzed experimental data to derive a fraction of PCBs that are picked up from concrete by dermal contact.

Massachusetts Department of Public Health: Prepared a public communications essays on what citizens can do to support improved air quality.

Medical Product Manufacturer: Prepared a risk assessment for air toxics produced during malfunction of a medical device used to assist breathing.

Michigan Occupational and Environmental Medical Association (MOEMA): Prepared and delivered a risk assessment tutorial for MOEMA's Continuing Education program.

Mining Company: Evaluated the epidemiological basis for the toxicity of arsenic in soils. Evaluated metals toxicity factors and site-specific bioavailability of metals. Calculated a cancer potency factor for arsenic, using cancer prevalence data in combination with water intake.

National Institute of Environmental Health Sciences -- Division of Research Grants: Participated in the Radiation Study Section Peer-Review Panel of grant applications on EMF Health-Effects Research.

Navy Occupational Health and Preventive Medicine Program: Prepared and delivered seminars and workshops to U.S. Navy medical personnel on the current research on the health effects of electric and magnetic fields (EMFs).

New Mexico Environmental Department: Prepared a health risk assessment for measured and modeled concentration of 80 airborne chemicals as detected or predicted for a suburb of Albuquerque, NM.

Refinery: Prepared a multipathway human health risk assessment for air emissions from a petroleum refinery. Our risk assessment preparation process was monitored by a Multi-Agency Task Force composed of regulators, educators, union members, and local officials.

School District on Long Island: Assessed possible environmental, occupational, and lifestyle risk factors for early-term miscarriage.

University of Denver: Analyzed the potential health impact of uranium disposal from munitions testing ("depleted uranium") as it was practiced in the 1960's and 1970's.

Uranium Mill: Evaluated the health implications of radioactive substance migration as predicted by different EPA and DOE models.

U.S. Department of Energy: Prepared a risk communication strategy for a nuclear test site where detonation of underground atomic devices had the potential to contaminate groundwater.

U.S. Department of Justice: Prepared an analysis of the health hazards of the Love Canal Superfund site (Niagara Falls, NY) as they were known at the time of the emergency declarations in 1978 and in 1981.

U.S. Department of Justice: Prepared a report and provided expert testimony on human toxicology with regard to soil contamination at a RCRA site.

U.S. Department of Justice: Prepared reports and provided expert testimony on asbestos, sulfuric acid, and airborne particulate inhalation toxicology.

U.S. Environmental Protection Agency: Analyzed the health risks of a remediation alternative at the Bloody Run Creek section of the Hyde Park Landfill superfund site (Niagara Falls, NY).

U.S. Environmental Protection Agency, Health Effects Research Laboratory: Assisted in preparing a database of non-cancer health effects for 189 Hazardous Air Pollutants.

U.S. Environmental Protection Agency, Environmental Criteria and Assessment Office: Participated in peer review group that evaluated research proposals on "Indoor and Ambient Air Risk Assessment Methodologies."

Utility: Analyzed the relationship between inhaled carbon monoxide concentration and blood carboxyhemoglobin. Performed sensitivity analysis on all the variables involved.

Waste Management Company: Evaluated health risks for a medical waste incinerator, including a multiple-pathway (ingestion, inhalation, dermal, mothers' milk) health risk assessment.

World Health Organization: Helped prepare a WHO research report on electric and magnetic field (EMF) health effects. Presented a lecture on EMF health effects at a WHO workshop in Geneva, Switzerland. Published review article on RF health effects.

Academic Research Projects *(abbreviated)*

National Heart, Lung, and Blood Inst.:	"Physical Determinants of Lung Function and Dysfunction."
National Heart, Lung, and Blood Inst.:	"Pulmonary SCOR: Chronic Diseases of the Airways."
National Cancer Institute:	"Magnetic Field Effects on Macrophages."
National Inst. of Environ. Health Sci.:	"Inhaled Particle Retention in Normal and Diseased Lungs."
National Heart, Lung, and Blood Inst.:	"Particle Location and Ingestion by Lung Macrophages."
National Inst. of Environ. Health Sci.:	"Factors Influencing Deposition of Inhaled Aerosols."

Publications – *Articles*

Valberg, PA; Van Deventer, TE; Repacholi, MH. 2007. "Base stations and wireless networks: Radiofrequency (RF) exposures and health consequences." *Environ. Health Perspect*. (in press).

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Abstracts & Reports *(list available on request)*

Invited Lectures *(past 8 years only)*

11/7/06 "What is EMF? How EMF Interacts with Organisms." Presented at the *Cyprus International Institute for the Environment and Public Health symposium on "Electromagnetic Fields: Sources, Health Effects, and Regulations,"* Nicosia, Cyprus.

6/19/06 "Pulmonary Deposition and Clearance of Particles." Presented in the course *"Comprehensive Industrial Hygiene: Practical Applications of Basic Principals,"* Harvard School of Public Health, Boston, MA.

5/18/06 "Health Hazards of Nanoparticles." Presented at *"A Mock Hearing: Environment, Health & Safety" at the NanoBusiness Alliance Meeting*, New York City, NY.

4/25/06 "Inhalation Risk Assessment: Extrapolating from Macro-materials to Nano-materials." *Overcoming Obstacles to Effective Research Design in Nanotoxicology*, Cambridge, MA.

10/6/05 Panelist for: "A Reevaluation of the Association Between Diesel Exhaust Exposure and Lung Cancer." *Air & Waste Management Association (AWMA) Specialty Workshop on "Diesel Exhaust,"* Chicago, IL.

6/20/05 "The Respiratory Tract as a Portal of Entry for Airborne Chemicals in the Work Environment." Lecture at the *Harvard School of Public Health course on "Comprehensive Industrial Hygiene,"* Boston, MA.

6/16/05 "Electromagnetic Fields, Base Stations, and Wireless Networks: Exposures & Health Consequences." *WHO Workshop, 15-16 June 2005, at the World Health Organization,* Geneva, Switzerland.

2/11/05 "Generation of Charged Aerosols by High-Voltage Electric-Power Lines." *American Association for Aerosol Research, Specialty Conference on Particulate Matter,* Atlanta, GA.

2/4/05 "Magnetic Microparticles Detect and Probe Cytoplasmic Motions." *Bioelectromagnetics Society Winter Workshop,* Phoenix, AZ.

6/21/04 "Pulmonary Deposition and Clearance of Particles." Harvard School of Public Health Continuing Education Course on *Fundamentals of Industrial Hygiene*, Boston, MA.

1/27/04 "Quantitative and Qualitative Factors that Determine Health Risk: Explaining Risk to Judges, Juries, and Communities." *Mealey's Water Contamination Conference*, Pasadena, CA.

9/14/02 "Health Effects of Air Pollutants." Annual Scientific Meeting of the Michigan Occupational and Environmental Medicine Association *Current Topics in Occupational and Environmental Medicine,* Frankenmuth, MI.

6/18/01 "Pulmonary Physiology, and Lung Deposition and Clearance of Particles." Harvard School of Public Health Continuing Education Course on *Fundamentals of Industrial Hygiene*, Boston, MA.

11/14/00 "Effects of Air Pollution on the Human Lung." Lecture in Tufts University course CEE 136, *Air Pollution*, Medford, MA.

7/26/00 "Review of Ambient Air Quality as it Relates to Proposed Emission Standards for Massachusetts Power Plants." *Testimony before the Massachusetts Department of Environmental Protection*, Boston, MA.

1/10/00 "Useful Concepts in the Physics of RF." *RF Safety: Science, Compliance and Communication*, Electromagnetic Energy Association and the University of Texas Health Science Center, San Antonio, TX.

12/16/99 "Exposure to inhaled pesticides and human health risks." *51st Annual Crop Protection School.* Office of Continuing Professional Education, North Carolina State University, Raleigh, NC.

7/21/99 "How do Endogenous Forces Compare to EM Forces and Torques on Electrical Charges and Magnetite?" *11th International Congress of Radiation Research,* Dublin Inst. of Technology, Dublin, Ireland.

6/7/99 "Lack of Concordance between Reported Lung Cancer Risk Levels and the Occupation-Specific Potential for Diesel Exhaust Exposure." *Third Colloquium on Particulate Matter and Human Health,* Durham, NC..

3/8/99 "Relative Risk Issues in Urban Pesticide Exposure and Children's Health." *Association of American Pesticide Control Officials,* AAPCO States/Industries Forum, Washington, DC.

1/13/99 "Panel Discussion on Health Effects of Wireless Technology." *Cape Cod Commission,* Deliberations at Cape Cod Community College, Barnstable, MA.

12/8/98 "Review of Health Issues in a Proposed Antenna Upgrade." *City of Newton Health Department*, Land Use Committee Deliberations, Newton, MA.

11/30/98 "Overview of radio wave health effects." Wayland, MA, Cellular Telephone Committee, Wayland Town Meeting Warrant.

8/3/98 "Exposure assessment in power-line-EMF and radio-wave epidemiologic studies." *EPE.215T Environmental and Occupational Epidemiology,* Harvard School of Public Health, Boston, MA.

4/22/98 "Health risks from electrical power lines and cellular telephones." *EH.202D Principles of Environmental Health*, Harvard School of Public Health, Boston, MA.

3/23/98 "Inhalation and Dermal Exposure to Occupational Chemicals." Harvard School of Public Health, Continuing Education Course on *Fundamentals of Industrial Hygiene*, Boston, MA.

Manuscript Peer Reviewer for the Following Research Journals

American Industrial Hygiene Journal; American Journal of Physics; American Journal of Respiratory Cell and Molecular Biology; American Review of Respiratory Disease; Atmospheric Environment; Bioelectromagnetics; Biophysical Journal; Biorheology; Cell Biophysics; Critical Reviews in Toxicology; Environmental Geochemistry and Health; Environmental Health Perspectives; Environmental Science & Technology; Epidemiology; Experimental Lung Research; Fundamental and Applied Toxicology; Hepatology; Human and Ecological Risk Assessment; Human and Experimental Toxicology; IEEE Biomedical Engineering; IEEE Transactions on Plasma Science; Journal of Aerosol Medicine; Journal of Applied Physiology; Journal of Applied Toxicology; Journal of Occupational and Environmental Hygiene; Journal of Occupational and Environmental Medicine; Nature; Nonlinearity in Biology, Toxicology, and Medicine; Radiation Research; Risk Analysis: An International Journal; Regulatory Toxicology & Pharmacology; Science; Tissue & Cell; USGS Environmental Geochemistry of Mineral Deposits (Reviews in Economic Geology series).

Exhibit B

Past 4 Years of Expert Testimony (Trial and Deposition) for Dr. Peter A. Valberg through January 15, 2007

Plaintiff	Defendant	Case #	Court	District	Date(s)
United States of America	Illinois Power and Dynegy Midwest Generation (*, D)	99-CV-833-MJR	Federal Court	District of Maryland	1/13/03
Danny Aguayo, *et al.*	Pacific Gas and Electric, *et al.* (*, D)	BC 123749	Superior Court	State of California, County of Los Angeles	6/19-20/03
United States of America	Ohio Edison *et al.* (*, D)	C2-99-1181	Federal Court	District Court, Southern District of Ohio	5/5/04
Larry and Martha Dampier	Norfolk Southern Railway Co. (*, D) and General Electric Co.	02-VS-027714-C	State Court of Georgia	Fulton County	3/11/05
Josephat Henry, *et al.*	St. Croix Alumina, LLC, Alcoa, Inc.; *et al.* (*, D)	Civil No. 1999/0036	District Court of the Virgin Islands	Division of St. Croix	4/7/05
United States of America	Westvaco Corporation (*, D)	Civil No. MJG00-2602	U.S. District Court	District of Maryland	4/22/05
United States of America	Westvaco Corporation (*, D)	Civil No. MJG00-2602	U.S. District Court	District of Maryland	6/8/05
United States of America	Westvaco Corporation (*, T)	Civil No. MJG00-2602	U.S. District Court	District of Maryland	8/25/05
Takeda Pharmaceuticals (*, D)	Mylan Pharmaceuticals, *et al.*	03-CV-8253	U.S. District Court	Southern District of New York	9/15-16/05
Robert D. Thornell, *et al.*	Cooper Farms, *et al.* (*, D)	C1 03 155	Common Pleas Court	State Court of Ohio, Paulding County	9/19/05
Takeda Pharmaceuticals (*, T)	Mylan Pharmaceuticals, *et al.*	03-CV-8253	U.S. District Court	Southern District of New York	1/18/06
Citizens Against Pollution	Ohio Power Co. (*, D)	2:04-CV-0371	U.S. District Court	Southern District of Ohio	1/23/06
Robert D. Thornell, *et al.*	Cooper Farms, *et al.* (*, T)	C1 03 155	Common Pleas Court	State Court of Ohio, Paulding County	2/23/06

* Party on whose behalf expert testimony was offered T = at trial D = at deposition